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Cancer Mortality in an Industrial Area of Baltimore[1]

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INTRODUCTION

Arsenic has long been known to be a poison when ingested in large quantities by man, animals, or plants. It is known that continued ingestion of high natural levels of arsenic in water or food will produce skin lesions including cancer (Tseng *et al.*, 1968; Braun, 1958). Consumption of arsenic as a therapeutic agent is also known to cause skin lesions (Neubauer, 1947).

The risk of inhalation of arsenic has not been as extensively investigated. Workers exposed to arsenic in the manufacture of pesticides have an increased risk of lung cancer and lymphomas (Ott *et al.*, 1974; Baetjer *et al.*, 1975). Less is known about the chronic health effects in the general population exposed to arsenic in the air. It is known that children around smelters may have high arsenic levels in nails and hair but it is not clear whether these observed indications of absorption of the agent also indicate long-term toxicity. Blot and Fraumeni (1975) have suggested that there may be an association between excessive lung cancer mortality and the existence of nonferrous smelting industries in several counties in the United States. It is not known whether some by-product of this industry such as arsenic is associated with these carcinogenic effects.

The purpose of the current study is to determine whether there is an excess mortality from cancer in the population which resides near a chemical plant in the inner city of Baltimore and whether any observed excess can be associated with previous exposure to arsenic. The plant produced insecticides, herbicides, and other arsenic products from 1897 until early 1976. In 1952, the original plant was torn down and a new one erected with better hygienic conditions for the workers. The plant produced arsenic acid, calcium and lead arsenate, Paris green (a cupric acetoarsenite), and sodium arsenite. In the past, all products were dried and packaged except sodium arsenite which was shipped as a liquid. Paris green was not produced after the early 1950s and no dry arsenicals after 1973. Other pesticides such as chlorinated hydrocarbons and organophosphates were not produced at this facility but were made into formulations on-site since 1947. There are

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several other industries which are currently located in the area or have been manufacturing in that vicinity in the past.

METHODS

The census tracts which were selected as having had possible environmental exposure to arsenic from the point source of the pesticide plant were defined empirically as those for which at least 50% of their area lay within a ¾-mile radius of the plant. This distance was chosen so that large tracts which lay across the river and in which the majority of the population did not reside within a 1-mile radius of the plant would not be included. The four index census tracts which fit these criteria were 2303, 2302, 2404 and 2301. The tract in which the plant was located is 2303.

The comparison group of census tracts consisted of all tracts which matched the index ones on age distribution, race, sex, and socioeconomic factors. Index tracts 2303, 2302 and 2404 were similar in these matching characteristics and were compared to the same set of comparison tracts designated as Match I. Index tract 2301 differed in age and race distribution from the others and was compared to a second set of tracts, Match II. Death rates for the index tracts in 1958 through 1962 were compared to tracts selected for matching through information available from the 1960 census. The matching criteria used to select control tracts for comparison of death rates in all other years were derived from the 1970 census. The age distribution differs in the tracts across time intervals, so to compare rates between years, the figures have been age-adjusted.

The initial matching criteria for the 1970 census were:

Age distribution ± 10% for each age,
Race ± 15%,
Sex ± 5%,
Median income ± $1000,
% below poverty level ± 10%,
% head of household over 65 years ± 20%.

The matching criteria based on the 1960 census were the same except that the variation in median income was reduced to reflect current inflation, and information on the last two characteristics was not available in the earlier decade.

The matching tracts in 1970 and 1960 are shown in Figs. 1 and 2. A total of 18 Match I control tracts was identified from 1970 census data and 45 tracts from 1960 data. A total of 5 Match II tracts was found in both census periods. The variation in the numbers of Match I tracts between the two periods is the result of changing racial distribution especially in middle income census tracts over the past 10 years. The 3 index tracts have a predominantly white population and fewer census areas have that racial distribution in the later time period.

The index area was stable with an increasing proportion of individuals living in the same household for 5 to 7 years from the 1960 to the 1970 census. This stability is also reflected in the slight increase in age of the population of the area.

The scattered distribution of the control tracts has placed them in areas which may also have had different risks. The adjacent tracts contiguous to the index ones

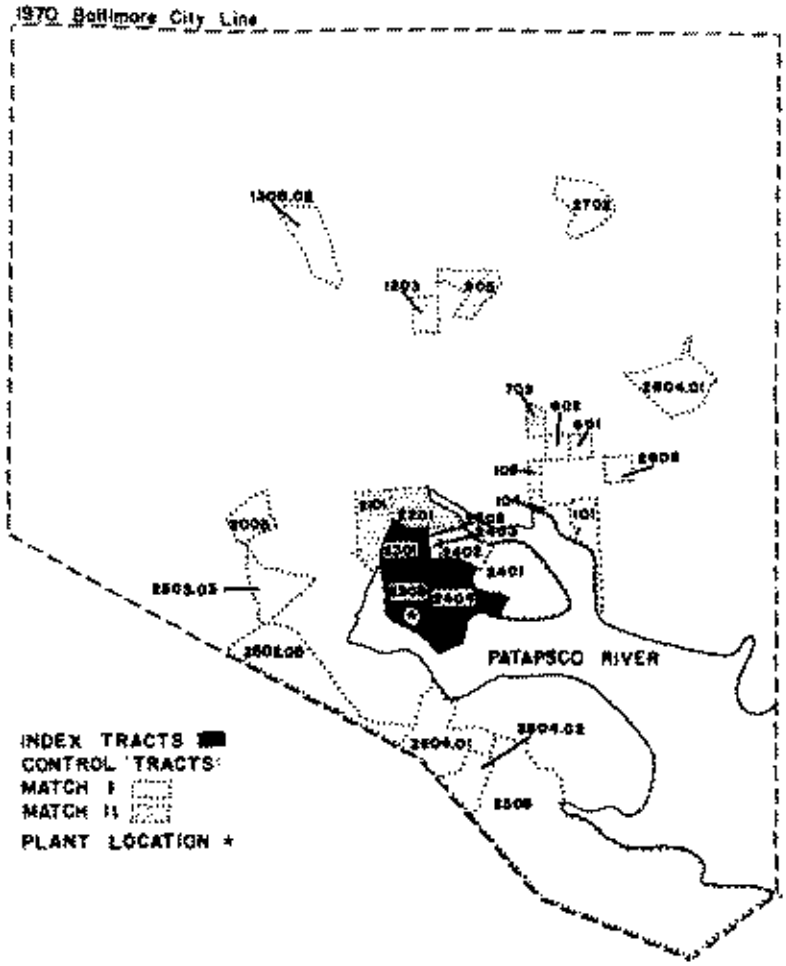

FIG. 1. Map of Baltimore City showing location of 1970 index and control census tracts.

may have had minimal exposure to the same agents as in the major area. Southern tracts are in heavy industrial areas as are the central tracts but the characteristics of the populations and their stability are different. The northern area consists of mainly residential dwellings with little industrial exposure. For these reasons the controls were divided into four groups for comparison—adjacent, south, central, and north.

Cancers were identified by examining all certificates of deaths which occurred within the city for the years 1958–1962 and 1968–1974. The death was selected for study if cancer appeared as a cause listed anywhere on the death certificate, with the exception of the years 1973–1974 where only cancers listed as underlying causes were chosen. Deaths of city residents were selected from the total cancer list. This procedure would not include the deaths of city residents which occurred

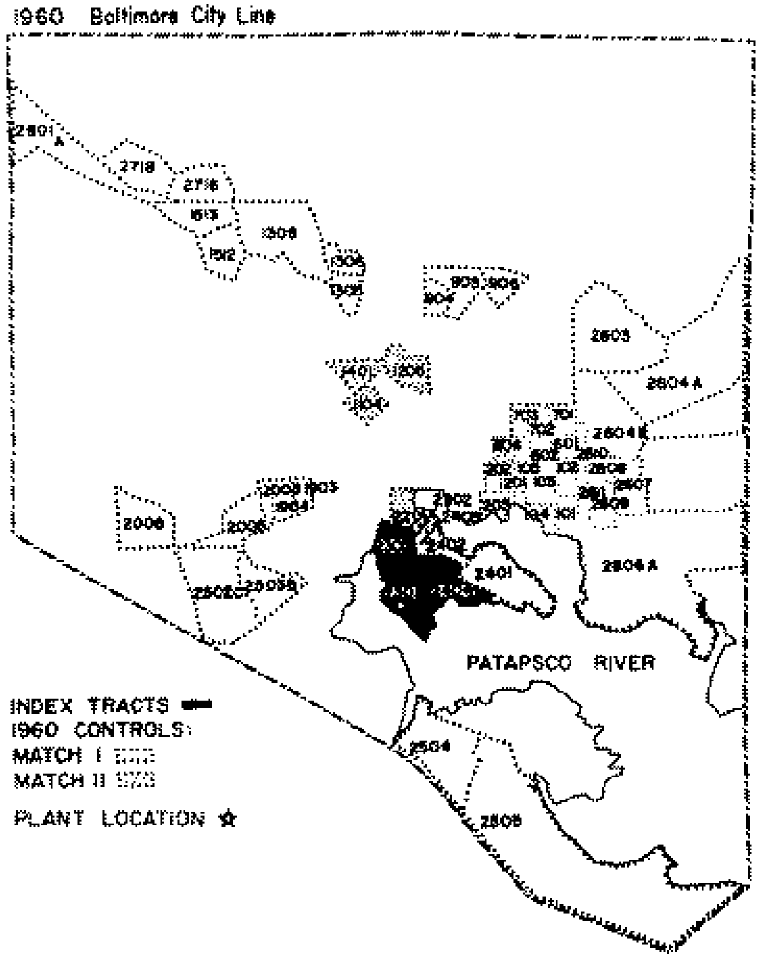

FIG. 2. Map of Baltimore City showing location of 1960 index and control census tracts.

outside the city. In order to determine the extent of these differences we abstracted information on out-of-city deaths of city residents for the 3 years, 1970–1972. The proportional increase in deaths for the index tracts was 5% and for the control tracts 13 to 15%. This difference is not large enough to account for the variation in cancer rates observed. Deaths were included only once using either underlying cause or first cancer listed. Adjustments were made in the changing codes in the seventh and eighth revisions so that data by site of cancer were compatible for the total period.

The hospital records of a sample of cancer deaths were reviewed to verify the accuracy of death certification of cancers in Baltimore, to identify any possible differences in diagnosis by area in the city, to determine any variation in pathological characteristics of cancers in index and comparison areas, and to investigate

differences in personal characteristics such as smoking as described in hospital charts. The review specifically focused on unusual cell types of lung cancer and possible arsenic-associated symptoms and diseases in cancer patients from the index and control areas.

The soil was sampled for the presence of arsenic in the areas near the chemical plant. The original selection of sampling sites was determined both by distance from the plant and by direction from north through south coordinates. We intended to collect about 50% of the samples within a ¼-mile radius and 40% of the next ¼-mile distance with the remaining samples collected further out on the radii. Control samples would be taken from two parks nearby but at a distance greater than 1 mile from the plant. The field survey team had problems adhering to the sampling design since the sources of soil were limited in the area. We attempted to take samples near residences whenever possible as long as there were no obvious problems of tree cover, water runoff, or redevelopment. For those few samples taken at private housing, the residents were interviewed concerning the use of herbicides or pesticides in the area and the sample was avoided if the soil had been treated. After collection of the original 101 samples taken at 35 sites under these directives and including additional samples taken in the park, a second set of samples was collected in a north and northwest direction to determine how far distant the high levels could be detected. Special emphasis was placed on sampling from the park which was adjacent to the plant. This park has a central grassed area which had recently been resodded. Surrounding the park was a dirt track which had been undisturbed. Part of this path was adjacent to the fence along the plant boundary and near the areas where railroad cars were filled. Another portion bordered on the water and the last was adjacent to railroad tracks.

Samples were collected at 1, 2, and 4 in. at each location unless otherwise noted. A core sampler, with a ¾-in. bore and marked at 1-in. intervals, was driven into the ground and samples were removed down to the appropriate depth marked. A 1-ft. circle was marked off around a selected site and a set of samples was collected according to the described technique until the 30-ml polyethylene sample bottle was filled with soil from the appropriate depth but from different core samples. Initially we had tested four sites using consecutive 1-in. samples down to a depth of 4 in. We found that samples at 3 in. were usually close to those at 4 and thus it was elected to take the extreme depth and discard the 3-in. level. Samples in control areas were all obtained from two city parks, Riverside or Federal Hill.

All instruments used in collecting samples were free of arsenic. The analysis was done using either conventional flame or flameless atomic absorption spectrophotometry depending on initial level of arsenic.

RESULTS

Mortality by Tracts

The crude rates for cancers at four specific sites, oral, pancreas, lung, and prostate as well as for all cancers are presented for males in Tables 1 and 2. The

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TABLE 1

AVERAGE ANNUAL CRUDE DEATH RATE PER 100,000 FOR INDEX AND CONTROL CENSUS TRACTS, MALES 1958–1962

	Match I													
	2303		2302		2404		Adjacent		South		Central		North	
Cause	No.	Rate	No.	Rate	No.	Rate	No.	Rate	No.	Rate	No.	Rate	No.	Rate
Oral	0	—	2	21.1	1	9.5	5	19.3	10	8.3	20	7.6	11	8.9
Pancreas	1	18.5	0	—	2	19.0	3	11.6	8	6.6	31	11.8	18	14.6
Lung	4	74.1	12	126.8	8	75.9	23	88.8	79	65.4	193	73.6	60	48.8
Prostate	2	37.1	2	21.1	3	28.4	3	11.6	19	15.7	43	16.4	23	18.7
All cancer	11	203.9	26	274.8	32	303.5	56	216.1	244	202.1	615	234.6	293	238.2

	Match II													
	2301				Adjacent				Central				North			
	White		Nonwhite		White		Nonwhite		White		Nonwhite		White		Nonwhite	
Cause	No.	Rate	No.	Rate	No.	Rate	No.	Rate	No.	Rate	No.	Rate	No.	Rate	No.	Rate
Oral	0	—	0	—	1	21.8	0	—	0	—	0	—	3	18.4	0	—
Pancreas	0	—	1	16.8	0	—	1	22.9	1	17.7	1	18.9	2	12.2	0	—
Lung	3	75.2	2	33.5	2	43.6	1	22.9	5	88.7	6	113.6	17	73.4	8	39.3
Prostate	1	25.1	2	33.5	1	21.8	1	22.9	0	—	0	—	5	30.6	3	14.7
All cancer	10	250.6	16	268.2	8	174.3	12	274.9	14	248.2	17	322.0	43	263.2	39	191.6

TABLE 2

AVERAGE ANNUAL CRUDE DEATH RATE PER 100,000 FOR INDEX AND CONTROL CENSUS TRACTS, MALES 1968–1974

							Match I Adjacent							
	2303		2302		2404				South		Central		North	
Cause	No.	Rate	No.	Rate	No.	Rate	No.	Rate	No.	Rate	No.	Rate	No.	Rate
Oral	2	33.9	0	—	0	—	4	12.7	7	6.8	6	9.0	5	11.7
Pancreas	2	33.9	0	—	1	8.0	3	9.6	9	8.8	9	13.4	3	7.0
Lung	18	305.0	12	103.3	12	96.1	24	76.4	111	108.3	60	89.6	48	112.4
Prostate	1	16.9	1	8.6	2	16.0	4	12.7	15	14.6	25	37.3	7	16.4
All cancer	33	559.2	26	223.8	24	192.2	86	273.9	254	247.9	206	307.7	110	257.7

							Match II							
	2301				Adjacent				Central				North			
	White		Nonwhite		White		Nonwhite		White		Nonwhite		White		Nonwhite	
Cause	No.	Rate	No.	Rate	No.	Rate	No.	Rate	No.	Rate	No.	Rate	No.	Rate	No.	Rate
Oral	0	—	1	16.9	2	20.5	2	20.3	1	17.5	0	—	0	—	1	6.9
Pancreas	0	—	3	50.7	0	—	3	30.5	2	35.1	0	—	0	—	1	6.9
Lung	7	113.3	13	219.5	13	133.1	16	162.6	4	70.1	5	102.8	15	124.5	6	41.3
Prostate	1	16.2	3	50.7	4	41.0	4	40.6	1	17.5	2	41.1	3	24.9	3	20.7
All cancer	17	275.0	27	455.9	34	348.2	42	426.7	17	298.0	13	267.2	41	340.3	28	192.9

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first table includes data for the 5-year period around the 1960 census and the second table for a 7-year period around the 1970 census. As can be seen the risk for lung cancer and for all cancers is excessive in the period around the 1970 census for tract 2303 compared to any of the control groups. This is not consistently true in the earlier period. The weighted relative risk of lung cancer in white males from index tract 2303 as compared to north controls which had the highest control rate is 2.5 as shown in Table 2 with a probability of 0.0005 as determined by the χ^2 calculated by the Woolf–Haldane method. The black males in tract 2301 also have a higher rate of lung cancer but this is not true for white males in the same tract. In the 1960 census period, although the lung cancer rate is higher in white males in tract 2303 than in the north and south controls, there is very little difference between the rates for all index tracts and for the adjacent and central controls. There are no differences in rates for males in tract 2301 and their control groups.

If we examine the comparable crude mortality rates for females in Tables 3 and 4, we can find no excess risk of cancer at any site for census area 2303. In fact, the overall cancer rate appears somewhat low especially in the 1958–1962 period. The mortality from breast cancer is slightly high in the early period and there are no deaths from cervical cancers. The lack of an observed increase in lung cancer in women in 2303 might be the result of a small population size. This will be discussed later in the report.

Adjusted Rates

The age distribution of the index tract changed with time and these differences were reflected in similar changes in the control group. In order to have appropriate comparisons the mortality rates for each cancer site and for all cancers have been adjusted using the method of standardized mortality ratios. The average annual Baltimore City mortality rates were calculated from all deaths in the 1968–1974 period and these values were used as standards to adjust the mortality in each time period. As seen in Table 5, the mortality ratios for white males in tract 2303 were high for cancers of the lung, pancreas, stomach, prostate, oral cavity, and all sites. The numbers of deaths except for lung and all sites were small but the pancreas cancer rate was still significantly higher than that for the city. White females in 2303 had an unremarkable overall cancer rate with excesses noted only for oral and rectal cancers of which only the latter ratio is significantly greater than unity.

In Fig. 3, we examine the lung cancer mortality in 2- or 3-year time intervals. Using rates adjusted by the direct method to the 1970 Baltimore City population as a standard, we find that the death rate for this cancer has always been higher in males from tract 2303 than from most controls but that it has been rising rapidly. The "all cancer" rates have also shown higher values than among controls. A preliminary look at the lung cancer rates for 1950–1951 indicated that the adjusted rates for that period were high for tract 2303 with a rate of 253 per 100,000 population as compared to rates ranging from 35.8 to 87.4 in other index tracts and controls.

Employees of an industry may live in close proximity and could have accounted for an increased mortality in the census tract due to occupational exposure. With

Prostate	3	16.2	3	50.7	4	41.0	4	40.6	1	17.5	2	41.1	3	24.9	3	28.7
All cancer	17	275.0	27	455.9	34	348.2	42	426.7	17	298.0	13	267.2	41	340.3	28	192.9

TABLE 3

AVERAGE ANNUAL CRUDE DEATH RATE PER 100,000 FOR INDEX AND CONTROL CENSUS TRACTS, FEMALES 1958–1962

Match I

	2303		2302		2404		Adjacent		South		Central		North	
Cause	No.	Rate	No.	Rate	No.	Rate	No.	Rate	No.	Rate	No.	Rate	No.	Rate
Oral	0	—	0	—	0	—	0	—	2	1.6	2	0.7	4	2.9
Pancreas	0	—	2	19.9	0	—	0	—	8	6.4	23	8.4	12	8.8
Lung	0	—	0	—	0	—	6	22.6	8	6.4	18	6.5	15	11.0
Breast	3	57.5	2	19.9	3	28.7	8	30.2	28	22.5	81	29.4	51	37.5
Cervix	0	—	1	10.0	1	9.6	3	11.3	15	12.0	34	12.4	12	8.8
All cancer	6	115.1	17	169.2	13	124.4	43	162.3	186	149.3	460	167.2	263	193.3

Match II

	2301				Adjacent				Central				North			
	White		Nonwhite		White		Nonwhite		White		Nonwhite		White		Nonwhite	
Cause	No.	Rate	No.	Rate	No.	Rate	No.	Rate	No.	Rate	No.	Rate	No.	Rate	No.	Rate
Oral	0	—	0	—	1	27.4	0	—	0	—	0	—	0	—	0	—
Pancreas	0	—	0	—	1	27.4	1	27.3	0	—	0	—	1	5.8	0	—
Lung	0	—	1	15.8	0	—	0	—	1	19.7	0	—	1	5.8	0	—
Breast	3	71.4	2	31.5	3	82.2	1	27.3	2	39.5	1	17.1	8	46.6	4	18.3
Cervix	1	23.8	1	15.8	1	27.4	1	27.3	2	39.5	1	17.1	5	29.1	3	13.8
All cancer	6	142.9	10	157.6	10	274.0	8	218.6	9	177.7	6	102.6	41	238.9	21	96.3

All cancer 6 [illegible]

TABLE 4

AVERAGE ANNUAL CRUDE DEATH RATE PER 100,000 FOR INDEX AND CONTROL CENSUS TRACTS, FEMALES 1968–1974

	March I													
	2303		2302		2404		Adjacent		South		Central		North	
Cause	No.	Rate	No.	Rate	No.	Rate	No.	Rate	No.	Rate	No.	Rate	No.	Rate
Oral	2	33.3	0	—	0	—	4	12.1	3	2.7	3	4.1	2	4.3
Pancreas	0	—	2	16.8	2	15.0	1	3.0	11	9.9	10	13.6	1	2.2
Lung	1	16.6	3	25.2	1	7.5	7	21.1	16	14.4	18	24.5	9	19.5
Breast	1	16.6	1	8.4	2	15.0	8	24.1	25	22.5	29	39.5	9	19.5
Cervix	0	—	3	25.2	0	—	3	9.1	6	5.4	5	6.8	0	—
All cancer	9	149.9	14	117.8	17	127.3	56	169.0	165	148.7	175	238.2	78	169.2

	March II													
	2301				Adjacent				Central				North	
	White		Nonwhite		White		Nonwhite		White		Nonwhite		White	
Cause	No.	Rate	No.	Rate	No.	Rate	No.	Rate	No.	Rate	No.	Rate	No.	Rate
Oral	0	—	0	—	0	—	1	9.7	0	—	0	—	0	—
Pancreas	1	16.4	0	—	1	10.9	2	19.3	1	16.7	0	—	3	23.0
Lung	2	32.8	1	15.4	2	21.9	7	67.6	1	16.7	1	18.1	3	23.0
Breast	5	82.0	3	46.2	6	65.7	4	38.6	2	33.4	0	—	8	61.4
Cervix	0	—	2	30.8	3	32.8	1	9.7	0	—	1	18.1	2	15.4
All cancer	15	246.0	18	277.1	26	284.6	25	241.5	14	233.6	5	90.5	34	261.1
													16	96.3

TABLE 5

TIME-ADJUSTED SMR'S BASED ON AVERAGE ANNUAL BALTIMORE CITY DEATH RATES, 1958–1962 AND 1966–1974

	Match I white males													
	2303		2302		2404		Adjacent controls		South controls		Central controls		North controls	
Cause	Obs.	SMR	Obs.	SMR	Obs.	SMR	Obs.	SMR	Obs.	SMR	Obs.	SMR	Obs.	SMR
Oral	3	2.45	2	0.88	1	0.43	9	1.43	18	0.88	29	0.91	16	0.89
Stomach	2	1.60	3	1.18	4	1.59	10	1.44	23	0.98	60	1.40	17	0.70
Colon	3	1.22	9	1.74	6	1.21	9	0.62	43	0.94	67	0.91	38	0.90
Rectum	1	1.09	3	1.62	4	2.19	6	1.18	14	0.85	34	1.25	15	0.97
Pancreas	5	4.15	0	....	3	1.31	7	1.09	21	1.01	43	1.27	21	1.10
Lung	25	2.74	30	1.72	23	1.31	57	1.19	206	1.35	282	1.23	118	0.93
Prostate	3	1.59	3	0.68	5	1.26	7	0.56	36	0.93	74	1.15	31	0.79
Bladder	0	—	3	1.38	2	0.97	5	0.85	20	1.03	37	1.11	28	1.45
Lymphomas	2	0.88	4	0.89	2	0.44	10	0.89	26	0.62	65	1.02	37	1.06
All cancer	54	1.94	67	1.21	63	1.15	168	1.10	543	1.10	893	1.13	423	0.96
	Match I white females													
	2303		2302		2404		Adjacent controls		South controls		Central controls		North controls	
Cause	Obs.	SMR	Obs.	SMR	Obs.	SMR	Obs.	SMR	Obs.	SMR	Obs.	SMR	Obs.	SMR
Oral	2	6.31	0	....	1	1.40	4	2.06	5	0.84	5	0.57	6	1.14
Stomach	1	1.32	3	1.74	0	....	5	0.96	14	0.89	37	1.30	18	1.01
Colon	1	0.45	7	1.36	5	0.94	13	0.83	41	0.89	81	1.01	40	0.82
Rectum	4	6.12	0	....	2	1.32	3	0.69	14	1.08	33	1.57	13	1.03
Pancreas	0	....	4	2.32	2	1.13	1	0.39	20	1.34	38	1.59	13	0.90
Lung	1	0.71	4	1.34	1	0.31	15	1.70	26	0.99	37	1.05	23	1.14
Breast	5	1.29	5	0.61	6	0.68	17	0.70	58	0.75	119	0.94	61	0.82
Cervix	0	—	5	2.30	3	1.27	6	0.97	25	1.13	43	1.14	13	0.61
Bladder	0	—	0	—	0	....	0	....	10	1.33	15	1.15	6	0.74
Lymphomas	1	0.65	4	1.19	3	0.86	6	0.61	36	1.22	52	1.05	24	0.83
All cancer	18	0.96	41	1.00	39	0.91	109	0.90	390	1.05	682	1.10	330	0.96

Note. Average annual Baltimore City rates (based on deaths in 1958–1962) were applied to the 1960 match population and were weighted for 5 years. Average annual Baltimore City rates (based on deaths in 1968–1972) were applied to the 1970 match population and were weighted for 9 years.

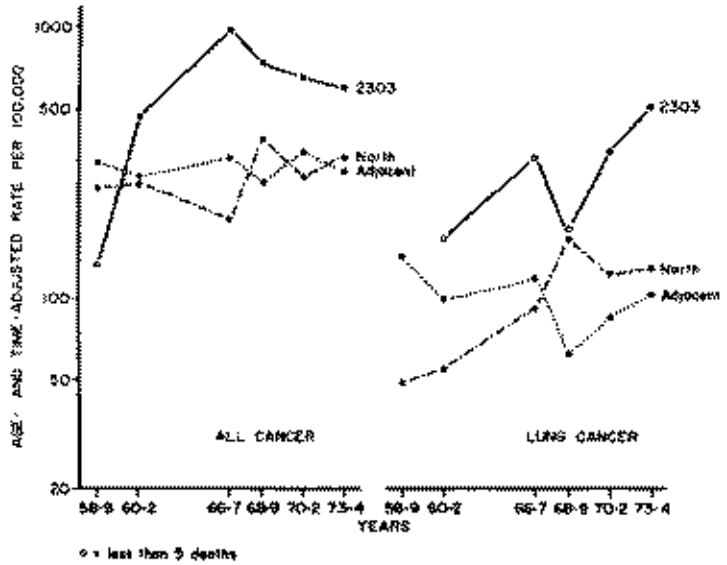

FIG. 3. Age- and time-adjusted rates per 100,000 for tract 2303 and North and Adjacent Controls. White males. (Semilogarithmic scale)

the cooperation of the company and the investigators studying the employees we reviewed lists of all employees to match with known deaths. Four employees were found among the cancer deaths in tract 2303 but removing these individuals did not change the significance of the rates.

The geographic distribution of cases was plotted on spot maps as shown in Figs. 4 and 5 for the two census periods. For both periods, lung cancer appears to be concentrated in an area about eight blocks wide and nine to twelve blocks long lying to the north and east of the plant. The area encompasses all of tract 2303 and parts of 2302 and 2301. If one takes all of tracts 2303, 2301, and 2302 which lie within a ¾-mile radius of the plant and calculates the proportion of lung cancers to all cancers in this area compared to the remaining census areas on Fig. 4, the proportion is 44.4% near the plant and 16.8% in the outer areas. For Fig. 5, the proportions show similar differences for 1973–1974 as in the previous 3 years. For the area within the defined census tract and ¾ miles of the plant, lung cancer represents 47.8% of the total cancers whereas in other areas it is only 33.3%. The northerly direction of this lung cancer excess is not compatible with the strong wind directions in that area. These winds arise in the northwest and west and should have carried contaminants to the east and southeast of the plant. The particles may have been moved by gentler winds and deposited nearby.

Soil Sampling

The highest arsenic levels are shown in Fig. 6. In most cases these levels occurred at 2 in. suggesting higher contamination in the past. Occasionally, as in the area adjacent to the plant, the levels were highest at 1 in. In general, arsenic

Cervix	9	....	2	....	2	....	0	....	10	1.33	15	1.15	6	0.74
Bladder	0	—	0	—	0	—	0	....	36	1.22	58	1.05	24	0.83
Lymphomas	1	0.65	4	1.19	3	0.86	6	0.61	390	1.85	682	1.18	350	0.96
All cancer	18	0.96	41	1.00	39	0.91	109	0.90						

Note. Average annual Baltimore City rates (based on deaths in 1958–1962) were applied to the 1960 match population and were weighted for 5 years. Average annual Baltimore City rates (based on deaths in 1968–1972) were applied to the 1970 match population and were weighted for 9 years.

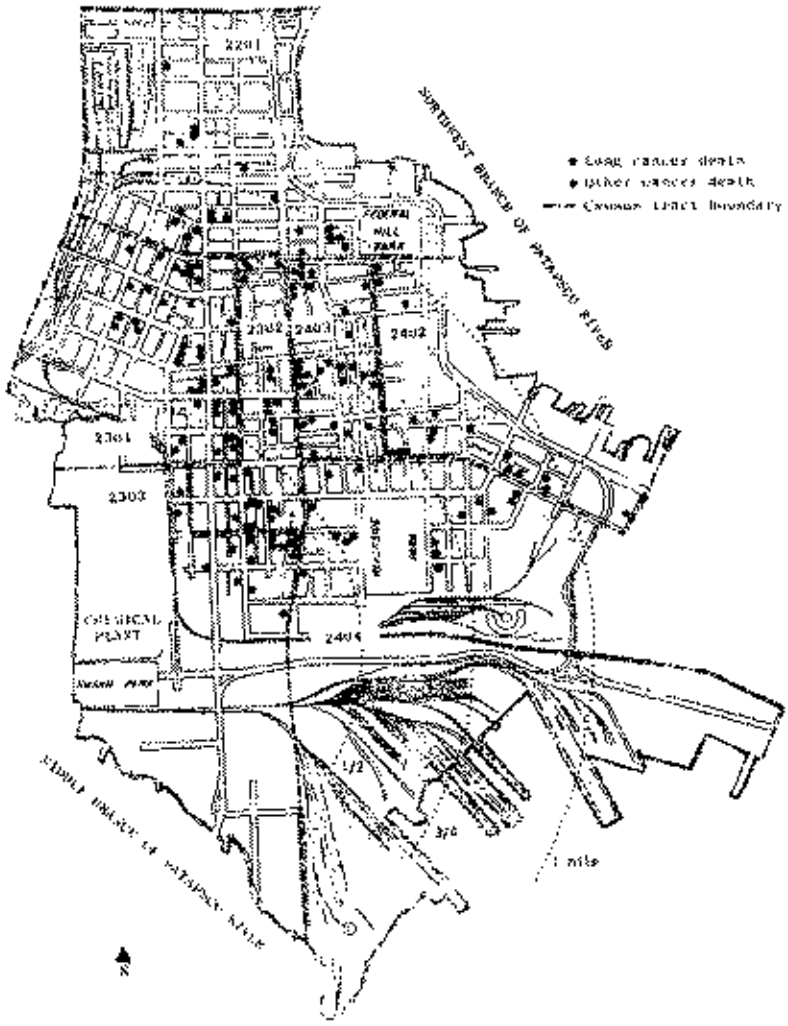

FIG. 4. Spot map showing cancer deaths for 1970–1972 by residence at death, excluding chemical plant employees.

levels were highest where lung cancer mortality was also highest. The mean arsenic level from 20 sample sites in tract 2303 was 63 ppm of arsenic. Even the omission of samples from the park adjacent to the plant only reduced the mean arsenic level to 38 ppm. Tracts 2301 and 2404 had means of 6 ppm and 2302 a mean of 4 ppm based on only 2 to 4 sample sites. All sites in the park had high levels except for an area which has been turned over and resodded and in which low arsenic levels were present. The 1-in. levels near the fence were as high as 695 and 226 ppm whereas at the opposite side of the park the values were only 29 to 97 ppm at 1 in. but as high as 46 to 161 ppm at 2 in. deep. From the soil levels of the

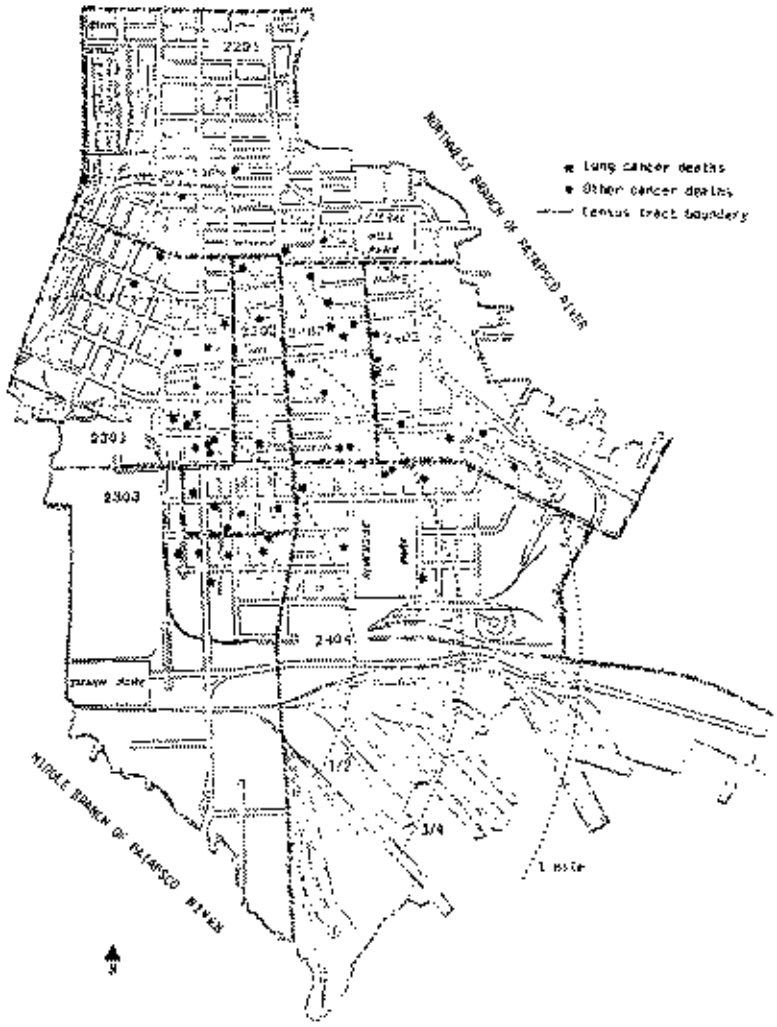

FIG. 3. Spot map showing cancer deaths for 1973–1974 by residence at death.

original samples, the data indicated high levels within a ½-mile radius of the plant. It was also apparent that higher levels were found in a northerly direction along the railroad lines.

Hospital Validation

The deaths of the total 14 years of study were included in the sample and stratified by control and index census tracts. The sample for hospital record review included all deaths for residents in the index tracts. Deaths for control tracts were stratified by age, race, and sex and three time periods, 1958–1962, 1966–1967, and 1968–1974. Four control deaths were selected randomly from

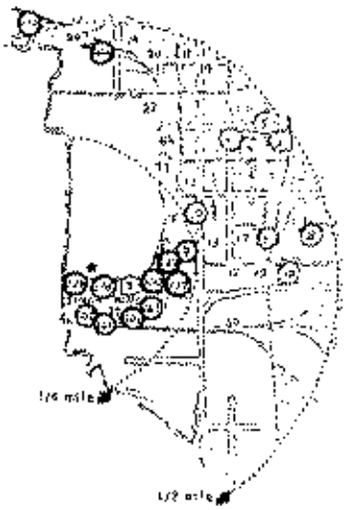

FIG. 6. Arsenic level in soil (ppm). Highest value at each site, summer 1976 and spring 1977.

each stratum for each index tract death within the same stratum. For the following analyses no attempt was made to expand the sample to the original population size.

The hospital abstract form included information on the following variables:

1. Cancer diagnosis;
2. Final diagnosis other than cancer;
3. Source of information for cancer diagnosis;
4. Arsenic-associated symptoms;
5. Personal characteristics such as smoking and occupation;
6. Description of pathological specimens, operative or autopsy findings.

Verification of the identification of the correct individual on the hospital record was done by name, birthdate, residence, and date of death.

Records were reviewed in eleven of the hospitals in Baltimore City. The remaining five noncooperating hospitals were small and did not limit substantially the number of records reviewed.

All possible medical conditions found on record review were listed and coded by the same nosologist who coded all the death certificates. The cell types were classified, in general, according to the "Manual of Tumor Nomenclature and Coding." Since this coding scheme does not appropriately classify the cells of several tumors, especially those of nonsolid origin, a revision of the scheme was made to include these cancers if we felt that their frequency was sufficient to warrant specific classification.

The causes of death were grouped into two time periods which represented the use of the seventh and eighth ICDA codes and grouped into causes as listed on the certificate by the first two digits of the code. These causes were then compared to the first four medical conditions or diagnoses as noted on the hospital records.

TABLE 6
LEVEL OF AGREEMENT BETWEEN DEATH CERTIFICATE CANCER CAUSE AND HOSPITAL DIAGNOSIS

	No.	%	
Complete Agreement (4 digits in ICDA code)	555	75.0	80.7
Agreement to 3 digits	42	5.7	
Agreement to 2 digits	31	4.2	
Metastasis entered on D.C. as underlying	13	1.8	
Multiple cancers on D.C., primary site not stated	41	5.5	
Other	18	2.4	
No cancer at autopsy or biopsy	20	2.7	
No records available	20	2.7	
Total	740		

The problem of validation of death certificate information was reviewed further by a physician who examined the data on the abstract forms. As indicated in Table 6, there was complete agreement in diagnoses to four digits in the ICDA code in only 75.0% of the cancer deaths. If classification to three digits only is used we can correctly verify 80.7% of the cases listed on the certificate. In 1.8% of cases metastatic lesions were identified on the death certificate as underlying and in another 5.5% multiple cancers were listed on the certificate and the primary site varied from that listed on the hospital record. There was no cancer diagnosis listed anywhere on the hospital record for 2.7% of deaths. A further examination of the method of diagnosis of cases was attempted in order to demonstrate whether differences in the methods might have changed the accuracy of death certification. Data from autopsy and histological examination of tissue were used for the diagnosis of 82% of the cases with complete agreement in records and 88.9% of cases where the agreement was less than perfect. Therefore, the consistency of cancer diagnosis on hospital records and death certificates is not related to the method by which the cancer was identified.

The method of cancer diagnosis differed only slightly in the larger hospitals with 69.7 to 91.4% of cases diagnosed by autopsy or histology. An examination of the differences in diagnosis by census tract has not been completed, but it is unlikely that there will be variations in results since we have included all hospitals used by individuals from the index area in the above evaluation.

An examination of the hospital records for possible arsenic-associated symptoms included gastrointestinal signs, skin lesions, Mee's lines on nails, neurological or neuromuscular symptoms, cardiovascular disease, stroke, and asthma. Only respiratory symptoms were slightly higher in the index tract but since there were so many records in which there was no comment about these symptoms, it is

TABLE 7
SMOKING HISTORY IN LUNG CANCERS FROM INDEX AND CONTROL TRACTS BY SEX

	Smoking		Nonsmoking		Unknown		Total
	No.	%	No.	%	No.	%	No.
Index tracts							
Male	30	59	2	4	19	37	51
Female	6	67	3	33	0	0	9
Control tracts							
Male	63	52	0	0	58	48	121
Female	5	22	0	0	18	78	23

difficult to interpret the small variation. We also sought information on diseases for which arsenic might have been used as a treatment, such as syphilis, trypanosomiasis, and amebiasis and the results indicated no higher frequency of these conditions among residents of index tracts.

Both smoking and drinking histories were abstracted from hospital records. Drinking habits were rarely recorded and smoking histories were also frequently missing. Table 7 indicates the smoking characteristics of lung cancer deaths in index and control tracts as determined from the hospital records. For 46.6% of the patients, the smoking histories are unknown. Despite that fact, we attempted to compare the smoking levels in index versus the control tracts. The percentage of smokers is slightly higher in the index tracts but the difference is not impressive. If one includes only those charts with a recorded history, almost all cases are positive for smoking in both index and control tracts.

The original hypothesis was that if arsenic had caused the lung cancers, the cell type of lesions from the index tract might differ compared to other areas with an expected predominance of small-cell or oat-cell tumors in the exposed tracts. The data in Table 8 would indicate that the cell types differ very little from index to control tracts.

DISCUSSION

An excess mortality from lung cancer has been demonstrated among men living

TABLE 8
CELL TYPES OF LUNG CANCER BY CENSUS TRACT

	Oat	Squamous	Adenocarcinoma	Epidermoid	Other	UK	Total
Tract 2303							
Male	2	8	—	1	1	4	16
Female	—	1	1	—	—	—	2
Other Index							
Male	3	18	2	1	1	8	33
Female	—	3	3	—	1	—	7
Control							
Male	12	57	16	4	3	18	110
Female	4	4	9	—	4	1	22

in a highly industrialized area of South Baltimore over a period from 1966 through 1974. The death rate is significantly higher than in control tracts in the later years.

The area surrounding the pesticide plant has high levels of arsenic in the soil which corresponds generally to the same areas where a high proportion of lung cancers to other cancers has occurred. There was no attempt to correlate directly arsenic levels to residences of lung cancer deaths.

The review of hospital records did not indicate that the excess of lung cancer deaths had occurred because of variations in diagnostic practices, cell types, or other factors. The information on other risk factors was poorly ascertained from hospital records.

There are some definite questions which arise in regard to the data. Why did the excess risk appear primarily in the late 1960s and early 1970s when the plant had existed and produced arsenical products since the early 1900s? The discrepancy could indicate that the plant did not account for the excess but some other local industry or occupational group accounts for the excess. It is also possible that the men in the area had a higher frequency of smoking and smoked a higher dose of cigarettes than did populations in the rest of the city. It is possible that selective mobility of younger, healthier males has left the area with a high risk among the remaining group.

The sudden rise in lung cancer might be related to the destruction of the old plant in 1952. Such an undertaking could have spread dust diffusely throughout the community. Under these circumstances we must ask why the concentration of lung cancer in the area does not coincide with the assumed wind spread of particles. It is necessary to examine further the mortality in the 1950 period to determine whether an excess existed at any time before the destruction of the old plant. It would be interesting to see if the appearance of the excess risk of lung cancer in the community coincided with that found in the workers within the pesticide plant. If we presume that arsenic may not be causing the excess then it would be necessary to examine the mortality experience of workers in other industries in the area, especially the natural gas plant, to see if they have an excess lung cancer mortality. In almost all cases, workers within an industry should have higher exposure and a greater risk of disease than the general public. It is necessary to investigate whether the increase in lung cancer can be related to a change in production or methods of operation of any of the businesses. For example, differences in handling arsenic, changes in formulation of pesticides, or the conversion of the gas plant from carburetted water gas to oil gas production could have created variations in level or type of pollution.

The fact that the excess lung cancer mortality has occurred only in men raises the question as to whether another environmental factor, differences in smoking characteristics, or occupation has caused the increased death rate in tract 2303. It is possible that smoking plus an environmental pollutant are required to produce the excess of cancer. The rates in older women then could be lower because they did not smoke and the possible synergistic effect of cigarettes and the environmental factor was not observed. Many of these questions might be determined by a community survey.

Further sampling for arsenic should be done to determine at what distance the

levels actually returned to background. It was first thought that the high levels along railroad lines might indicate a relationship to previous coal use. However, further investigation showed that the arsenic content of coal in local use did not reach levels as high as those measured along the tracks. Rail transport of materials from the plant may have been related to the high levels. Further investigation of this possibility is needed. It appeared that use of herbicides did not explain the arsenic levels in the rail beds.

In summary, men living in close proximity to a chemical plant which produced arsenicals have a higher risk of lung cancer than comparable individuals in other areas of the city. The distribution of arsenic in the soil near the plant and along the railroad line is higher than in control areas.

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DISCUSSION

J. STERBINOS: Out of curiosity, I would like to ask one question. As I recall the cementing of back yards in Baltimore was a public health measure to control rats. I believe it was done in the 1940s.

G. MATANOSKI: Some were done in the forties. In the 1950s, however, residents began to become discouraged with the lack of vegetative growth in their yards and many homeowners in the South Baltimore area put in cement even in areas that were not rat infested.

J. STERBINGS: Do you think that reduced the exposure to arsenic?

G. MATANOSKI: The exposure to arsenic probably was coming from the air, rather than from moving backyard dirt around. Of course, they might have had increased dust exposure by cleaning their cement areas.

J. LYON: Just one comment on the proportion of cancer death for which you can find no hospital record. That is a standard proportion which you see in registries covering populations. For about 1.5 to 3% of the cancers identified by death certificates you can find no hospital or at best only an emergency room record if they died in that facility. So I am not too impressed at that finding. It is quite consistent among cancer registries.

R. ALTMAN: Why do you think there was no lung cancer outbreak in females if you are attributing the risk to the ambient air?

G. MATANOSKI: One of the postulated explanations is that one had to be a smoker as well as having arsenic exposure to show the risk, thus the synergistic effect rate of lung cancer is higher in that area even though the smoking rates by

hospital record are the same because of the additional exposure to arsenic. Women in that area during that time and in that age group were probably not smokers.

R. ALTMAN: Wouldn't you expect that even if the risks were low in women you would anticipate some increase compared to other women?

G. MATANOSKI: I would have expected it, yes. However, I can't explain the excess mortality in men on any of the known risk factors such as occupation or smoking with the data which we have now. The conclusion may be different if we complete the proposed study and check out these data. They are the only other factors which I could think of that would make a man more susceptible to lung cancer from the environment.

R. ALTMAN: Is there any excess mortality of lung cancer in the workers in this plant?

G. MATANOSKI: Yes, that is why we originally started the community study. There had already been a report of an excess in other chemical plants. Investigators looked at the retirees in the plant, as commissioned by the industry, and they found excesses of lung cancer and lymphoma among their workers. A second study of all workers has been completed, but some information may be restricted at the present time. Reports from a thesis related to this work indicated an excess of lung cancer in male workers.

P. BUFFLER: With regard to the male excess, in addition to the review of employee records for the one company, could you get full occupational histories on the cases in that tract?

G. MATANOSKI: No, we wanted to do a community survey to get full occupational histories. All the information which I had was the occupation listed on the death certificate and on the hospital record at the time of diagnosis.

P. BUFFLER: The possibility still exists that there could have been some occupational exposures in that group?

G. MATANOSKI: Something that was different in that census tract compared to others? Yes. The one which I thought of was shipbuilding, because the residents live not too far from Maryland Drydock, a shipbuilding and repair operation, and also a high proportion of all our Baltimore City population works at Bethlehem Steel. When I actually looked at the hospital histories, there was no suggestion that that was true. There was a higher proportion of these occupational groups represented in the other areas. That could be because Bethlehem Steel represented high rates of employment histories in the eastern area of the city nearer the industrial facility.

A. BAHN: Were you able to get residential histories to determine whether these people lived in the area for a long period of time? And, secondly, you were saying something about the hoppers? Was arsenic carried in open hoppers or on boxcars with spray onto the track? Was that a possibility?

G. MATANOSKI: It could be dumped into a hopper car and be carried out through the rail lines. The spill could have blown off the car on the entire length of the line and that may be why it is higher near the railroad lines. It is the only reason we can find at the present time for the distribution of arsenic along rail lines. It can't be explained by coal dust or herbicides according to railroads. In

answer to your question about residence, no, we do not have a complete residence history. We have the residence at the time of hospitalization which in most cases, especially lung cancer cases, was the same as the one they had at death. There were rarely any differences unless they resided at the home of a relative. However, these are the only two points of residence which we have. One of the things we need is a community survey of resident histories. We need to survey the next of kin of these deceased individuals to find out if they lived in the area long enough to have had their cancer from exposure in that place.

P. LANDRIGAN: Do you have any data on ambient concentrations of asbestos?

G. MATANOSKI: Asbestos. No, I don't, not in that area.

P. LANDRIGAN: You mentioned just now that a shipyard was near the area. I was wondering whether it was near enough to have possibly caused some effluent effects within the general atmosphere in the area?

G. MATANOSKI: I don't know how far asbestos would go. The shipyard may be about 6 miles away.

P. LANDRIGAN: Oh, it's not that close.

G. MATANOSKI: It's not. It's across the bay, part of the bay on the other side, but it is close enough for them to get there so they work there.

G. TOKUHATA: Those 740 cases of cancer for which you have checked the diagnoses you said 80% agreed between the hospital records and the death certificates. Did you find any difference in this pattern between cases in the various areas? Are they the same or did you find any difference?

G. MATANOSKI: We didn't actually look for differences by area, we looked by hospital. A hospital usually services different areas. Perhaps, we should go back and look by area. However, we found no difference by hospitals, so I gave up further analysis at that point. The hospitals were absolutely the same across the board, with differences of no more than about 1 or 2% regardless of the institution.

UNKNOWN: Did you say the plant had ceased operation?

G. MATANOSKI: Yes, they put a bridge up in the area.

S. MILHAM: We did the same kind of study around the Tacoma smelter and again, we found a male excess only. However, it washed out when you took the workers out. They found a lot of arsenic in the soil, 2000 part per million, about a half-a-mile away. We know the arsenic has been there for a long time. The smoker–arsenic interaction hypothesis washes out because they checked it out in the smelter. The smokers actually had lower lung cancer rates for given arsenic concentration. So, I don't know what is going on!

G. MATANOSKI: We have another theory about the men but it is only a guess. The men down in that area play ball in the three fields. Only men play ball usually. It is possible that local boys from the general South Baltimore area have played ball there and lived there. This is at least one other activity that differs between men and women from that area.